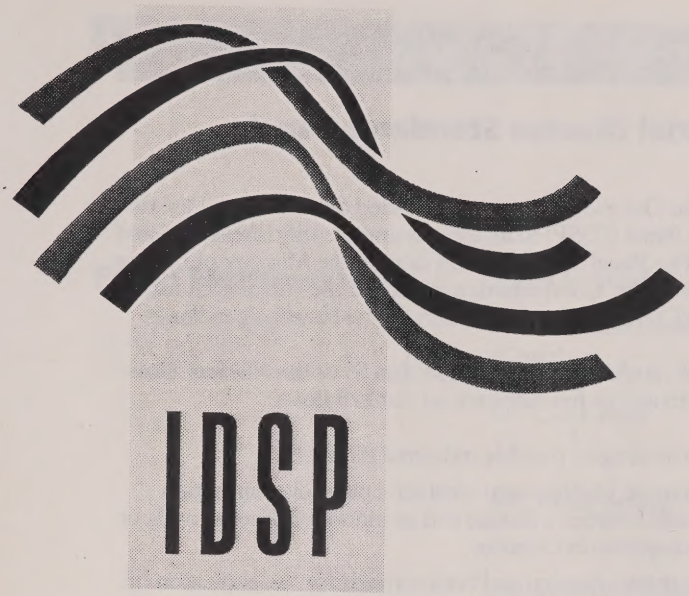
Interim Report to the Workers' Compensation Board on Aluminum

May, 1992

Industrial
Disease
Standards
Panel



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May, 1992

Industrial Disease Standards Panel
IDSP Report of Findings No. 9
Toronto, Ontario

Industrial Disease Standards Panel

In 1985 the Ontario legislature established the Industrial Disease Standards Panel (IDSP) to investigate and identify diseases related to work. The Panel is independent of both the Ministry of Labour and the Workers' Compensation Board. At the end of each fiscal year the WCB reimburses the Ministry for the Panel's expenditures.

The Panel's authority flows from section 95 of the *Workers' Compensation Act* and its functions are set out as follows:

(8) (a) to investigate possible industrial diseases;

(b) to make findings as to whether a probable connection exists between a disease and an industrial process, trade or occupation in Ontario;

(c) to create, develop and revise criteria for the evaluation of claims respecting industrial diseases; and

(d) to advise on eligibility rules regarding compensation for claims.

Decisions of the Panel are made by its members who represent labour, management, scientific, medical and community interests. Once the Panel makes a finding, the WCB is required to publish the Panel's report in the Ontario Gazette and solicit comments from interested parties. After considering the submissions the WCB Board of Directors decide if the Panel's recommendations are to be implemented, amended or rejected.

To assist with its work the Panel has a small staff of researchers, analysts and support people. In addition to its own staff, the Panel relies heavily on the advice of outside experts in science, medicine and law, as well as input from the parties of interest.

Canadian Cataloguing in Publication Data

Main entry under title:
Industrial Disease Standards Panel's Interim report to the Workers' Compensation Board on aluminum

(IDSP report, ISSN 0840-7274 ; no. 9)
Includes bibliographical references.
IBSN 0-77299853-1

1. Aluminum—Physiological effect. I. Ontario, Industrial Disease Standards Panel. II. Series.

RA1231.A5152 1992 615.9'25673 C92-092559-6

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Panel Members	*Appointment*
Ms. Nicolette Carlan (Chair)	May 16, 1991 to May 15, 1994
Dr. Carol Buck	June 1, 1991 to June 16, 1994
Mr. James Brophy	January 23, 1992 to January 22, 1995
Mr. William Elliott	November 7, 1991 to November 6, 1994
Mr. David Leitch	November 7, 1991 to November 6, 1994
Mr. John Macnamara	November 7, 1991 to November 6, 1994
Mr. Homer Seguin	May 28, 1989 to May 27, 1992
Dr. Michael Wills	November 7, 1991 to November 6, 1994

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Report #9

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Ministère du Travail de l'Ontario

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June 3, 1992

Mr. Odoardo DiSanto
Chairman,
Workers' Compensation Board
2 Bloor Street East
Toronto, Ontario
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Dear Mr. DiSanto,

Enclosed is a copy of the Panel's "Interim Report to the Workers' Compensation Board on Aluminum".

I would be pleased to discuss the Report with you. Please let me know when it would be convenient to do so.

Sincerely,

Nicolette Carlan,
Chair

encl.

The Issue and How it Arises

Boeing of Canada, deHavilland Division (now known as deHavilland Inc.) manufactures aircraft and McDonnell Douglas Canada Ltd. manufactures aircraft wings composed of aluminum. The workers at both plants have been very concerned about their exposure to aluminum dust and aluminum welding fumes.

The Workers' Compensation Board has no policy about aluminum claims. On December 16, 1988, the following questions were referred to the Industrial Disease Standards Panel by Dr. R. G. Elgie, then Chair of the Workers' Compensation Board:

> ***1) Is there scientific evidence that occupational exposure to aluminum constitutes a health risk to workers?***
>
> ***If the answer to this question is yes, then I am asking the Panel to address the following questions:***
>
> ***2) Are serum aluminum levels a good indicator for predicting the occupational health risks?***
>
> ***3) Under what conditions should a worker be removed from occupational exposure to aluminum?***
>
> ***4) What criteria should be used in the adjudication of claims arising from the adverse health effects? (49)***

After conducting some preliminary investigations, the Panel has decided to expand the scope of question 2) and ask:

> ***Are urine aluminum levels or other measuring methods useful as indicators for predicting occupational health risks?***

The Canadian Auto Workers have asked the Panel to recommend that the "precursor clause" in Section 1 (1) (n) (iii) of the *Workers' Compensation Act* be applied when certain blood aluminum levels are found.

Investigations by the Panel

The Panel has reviewed the medical literature about aluminum and its effects. The IDSP issued a Progress Report on October 20, 1989 which included a detailed literature review. (146) That Report identified the need for further investigations "concerning health effects among Ontario aircraft workers resulting from exposures to a variety of substances." (45)

Since then, the Panel has further reviewed the medical literature and has consulted several leading experts on the subject.

The Panel has reviewed Ministry of Labour hygiene reports on plant conditions at McDonnell Douglas made between 1985 and 1992 and at Boeing–deHavilland made between 1984 and 1992. Some of the current Panel members have visited both plants.

The Panel obtained an independent legal opinion on the precursor clause from the law firm of Cornish Roland. (20) The Panel has also considered a legal opinion provided by the Canadian Auto Workers. (16)

The Panel has solicited and considered several proposals for a study of the health effects of aluminum in these workers and has received comments on those proposals from several peer reviewers.

The Panel has routinely consulted labour and management involved in these issues.

Submissions by the Parties

The aircraft workers are represented by several locals of the Canadian Auto Workers. Union leaders state that there is sufficient evidence to establish that the workers' occupational exposure to aluminum could lead to serious health effects. They are particularly concerned about neurological illness and cognitive deficits. They state that some of the members are suffering from symptoms such as dizziness, headaches, nausea, memory loss and irritability. They attribute these problems to aluminum exposure because:

> Our understanding is that it is established that aluminum exposure in industry can and has caused neurological damage, lung disease and death. There is a great deal of evidence of aluminum related toxicity from animal research and the effects on in-vitro tissues. Further, the effect of aluminum intoxication on patients undergoing renal dialysis establishes that aluminum does cause neurological damage. And finally, it has been clearly established that people with Alzheimers have high levels of aluminum in the brain compared to the average person who has very low levels. (36)

The Panel's preliminary investigations found that the following evidence might support these concerns:

- Very high levels of aluminum in some kidney dialysis patients are the recognized cause of a usually fatal brain disorder known as dialysis encephalopathy syndrome ("DES");

- Encephalopathy has been reproduced in animals by exposing them to aluminum;
- There is considerable evidence that high levels of aluminum appear in the brains of victims of Alzheimer's Disease;
- Some, but not all, environmental studies have shown a greater incidence of Alzheimer's Disease in localities which have high levels of aluminum in drinking water; and,
- A preliminary report suggests that some northern Ontario miners who were exposed to high levels of aluminum may have suffered some cognitive effects as a result of that exposure.

Because of its concern for the workers' health, the Union has arranged for the Ontario Workers' Health Centre ("OWHC") to conduct medical monitoring of the workers in both aircraft plants. Based upon research conducted at the University of Western Ontario, the Union and the OWHC contend that "normal" aluminum levels in healthy, non-occupationally exposed persons are at most 371 nanomoles ("nmol") per litre of blood and 556 nmol/litre of urine. (73)

The OWHC's April 6, 1989 report states that, of 1029 McDonnell Douglas workers tested, 184 had at least one blood aluminum measurement above that normal level and when averaged by the number of tests per worker, 146 had blood levels above normal. The vast majority were under 500. However, there was one result at each of the following three levels: between 2601 and 2700; between 2001 and 2100; and between 1401 and 1500 nmol/litre of blood.

Although specific urine test results were not listed in that report, it does state that 459 McDonnell Douglas workers had at least one urine or blood level which was above the levels which they consider normal.

While the Union acknowledges that any "dose-response" relationship is not yet known (36), it maintains that there is sufficient evidence to establish that these levels increase the risk of damage to health and have expected that action would be taken to prevent damage to the health of these workers.

On this evidence, the Union is asking that workers who report blood aluminum levels in excess of 371 nmol/litre be removed from the workplace and compensated for lost time. In its view, the Workers' Compensation Board has the responsibility and authority to take this action under the "precursor clause" (Section 1 (1) (n) (iii)) of the *Workers' Compensation Act.*

The Union has also requested a comprehensive medical and epidemiological investigation of the health effects of aluminum exposure in the aircraft industry.

McDonnell Douglas made a detailed submission to the Panel (103) which included copies of scientific studies and several "Health and Safety Notes" published by the Company which contend that aluminum exposure at the plant is not a health hazard to its workers. The package also contained reports on blood aluminum tests indicating that, of 54 current employees tested, only one had a blood aluminum level above 372 nmol/litre. However, of 152 newly hired workers, 19 had blood aluminum levels above 372 nmol/litre on pre-employment tests. Of 58 recalled employees, 18 had results above that level before returning to work. McDonnell Douglas argued that aluminum levels are commonly raised by diet and the use of antacids and can be reduced to normal levels by controlling those sources regardless of occupational exposure.

Boeing–deHavilland has not made submissions to the Panel on the aluminum issue.

Although both companies were also asked to provide their comments on the appropriate application of the precursor clause, neither has done so.

The Panel's Findings to Date

1) Is there scientific evidence that occupational exposure to aluminum constitutes a health risk to workers?

The Panel has reviewed the medical literature on this subject and has consulted numerous experts in order to investigate a broad range of possible outcomes of aluminum exposure, including Alzheimer's Disease. It has reviewed epidemiological surveys of the aircraft, pyrotechnics and aluminum industries. It has also considered reports of clinical findings, research on animals, anecdotal evidence from workers, employers and doctors, and has reviewed WCB claims.

Background

Aluminum is a naturally occurring substance and is the most common metal in the earth's crust. (65, 92) Daily exposure is unavoidable by ingestion through food and water and by inhalation from the atmosphere. Aluminum has no known nutritional benefit. (134)

Normal daily intake in adults is estimated to be about 9 to 14 milligrams (120) but may be much higher depending on diet and intake of aluminum-containing medication such as antacids. (84, 65, 142) Normal total body aluminum content is in the range of 30 to 40 milligrams. (5)

*It is important to distinguish between the amount of aluminum to which people are **exposed** and the amount of aluminum which is not eliminated but is **absorbed** by the body and accumulated in tissue.*

All the medical experts consulted by the Panel have advised that it is important to distinguish between the amount of aluminum to which people are *exposed* and the amount of aluminum which is not eliminated but is *absorbed* by the body and accumulated in tissue.

In healthy people, the "blood-brain barrier" almost completely prevents aluminum from accumulating in the brain.

The skin, lungs and gastrointestinal tract "serve as almost complete barriers to aluminum absorption." (5) In healthy people, the "blood-brain barrier" almost completely prevents aluminum from accumulating in the brain. (23) Some researchers believe that Alzheimer's Disease involves a defect in the blood-brain barrier, which is discussed further below.

The highest accumulation occurs in the lungs and bone. (134) Although the concentration of aluminum is highest in lung tissue, 50% of body burden occurs in bone because bone mass is so much greater than lung mass. (58) Accumulation in the lungs (65) and brain (134) appears to increase with age. (32)

Occupational Surveys

Studies of overall death rates in aluminum workers found either no increase or lower rates than expected.

Mortality
Studies of overall death rates in aluminum workers found either no increase (47, 60) or lower rates than expected. (106, 59)

Cancer
A survey of the aircraft industry found that numbers of cancer and nervous system diseases were lower than expected. (59) In addition, a 1988 review of the literature concluded that, except perhaps in cases of extremely high exposure, aluminum is not carcinogenic. (91)

Two surveys of mortality in aluminum reduction plant workers found increases in cancer rates (128, 106) one of which attributed this to coal tar pitch volatiles. (106)

Two epidemiological studies found evidence of increased rates of bladder cancer in workers in the aluminum industry. (149, 147) One of those researchers related the increase to polycyclic aromatic hydrocarbon exposure (149) and the other related it to exposure to coal tar pitch volatiles. (147)

Respiratory effects
For a few years there were reports of lung fibrosis having occurred in workers who produced fine aluminum powder in the pyrotechnics industry. (110, 83, 111, 105) The cause of those diseases is now attributed to the mineral oil coating which was applied to the aluminum particles in England between 1948 and 1955. In those cases which did not involve coated aluminum particles, the cause of the fibrosis has been attributed to the workers' exposure to silica, rather than to their exposure to aluminum. (41, 112)

However, lung fibrosis in one Belgian metal polisher was attributed to the worker's heavy aluminum dust exposure. (40)

Two surveys found increased rates of respiratory disease in aluminum workers. (128, 60) In one of these, coal tar pitch volatiles were considered the cause. (60) Slight impairment in lung function has been observed (90), as has bronchitis, which was considered related to ozone exposure. (139) Wergeland and colleagues found increased rates of asthma (158) but some authors attribute the cause of asthma and bronchitis in such workers to high concentrations of mixed dusts. (112)

Another research team found no such increase in lung illnesses. (14)

There are reports of reduced lung function proportionate to workers' exposure to mixed dusts including aluminum. (150) Nine workers in an abrasives plant with "prolonged heavy" exposure to aluminum oxide dust were found to have pulmonary fibrosis. The cause was determined to most likely be exposure to aluminum oxide, but possibly exposure to mixed dusts. (82)

Neurological effects

A 1989 search of the Swedish Occupational Cancer Registry database for evidence of aluminum-related or neurological illness or symptoms in the Swedish aircraft industry found only one report which involved solvent exposures. (70)

A study of the neurologic health of its workers was conducted for Intalco Aluminum Corporation in the state of Washington because nine workers filed claims for neurological symptoms which they related to aluminum dust exposure. A small increase in self-reported neurological symptoms was found among the most highly exposed but no objective medical or neuropsychologic evidence of a condition which was common to all of them was found. Six workers displayed no objective medical findings on tests nor upon examinations and the remaining three had other unrelated health conditions which were thought to explain their symptoms. (165)

The Panel received documentation from the University of Pittsburgh which indicated more than the number of expected deaths from neurological disorders. Of 21,800 workers studied, seven deaths from neurological illnesses were found. (129) The number of cases is so small that any analysis of these figures is unreliable.

No Alzheimer's Disease or lung fibrosis deaths were found in a mortality study of 5,406 Canadian aluminum smelter workers.

Alzheimer's Disease

Gibbs found no Alzheimer's Disease nor lung fibrosis deaths in a mortality study of 5,406 Canadian aluminum smelter workers. (60)

French found no difference in the occupations of Alzheimer's Disease patients compared to controls but did find more head injuries in Alzheimer's victims. (57)

There is one report of Alzheimer's Disease occurring in one man who had worked for thirty years as an aluminum refiner in Japan. The authors compared his brain aluminum content to that of another Alzheimer's Disease victim and a victim of subacute sclerosing panencephalitis ("SSP"), neither of whom had occupational exposure to aluminum. They found aluminum accumulation in the aluminum refiner but not in either of the non-occupationally exposed victims. The authors concluded that aluminum does not necessarily contribute to the formation of Alzheimer's lesions, but it accumulates as a secondary effect in Alzheimer's victims who have had occupational exposure. (85)

Other

Between 1980 and 1987, the U.S. National Institute for Occupational Safety and Health (NIOSH) conducted health hazard evaluation surveys of eleven American aerospace, aircraft and aluminum-using operations. In one aerospace plant, aluminum welding fumes were found to be the cause of upper respiratory and eye irritations. In an office building, aluminum filings were likely responsible for neck, hand and facial rashes but no other health effects related to aluminum were found. Neurological symptoms were not a complaint so they were not investigated. (69)

Complaints of eye and respiratory tract irritation, skin rash and neurobehavioural responses prompted an investigation of health problems in a Washington state aircraft plant. The most probable cause of these symptoms, including neurobehavioural and autoimmune responses, was exposure to phenol formaldehyde resin and resin by-products. (10)

Aluminum and Encephalopathy

Dialysis Encephopathy Syndrome (DES) in kidney patients is believed to be caused by extremely high aluminum exposure.

It is known that aluminum in very large amounts can be neurotoxic. "Dialysis Encephalopathy Syndrome" (which is also called "DES" or "dialysis dementia") is a usually fatal condition which has occurred in some of the kidney dialysis patients whose blood levels rose to 7400 nmol/litre or above when their dialysis fluid contained large amounts of aluminum. (2, 24, 135, 65, 163) DES has now been largely eliminated because current treatment protocol requires removal of aluminum from dialysis fluid. (58)

The clinical features of DES are: stuttering; unclear speech; inability to speak; loss of ability to express, name things or recall names; persistent repetition of the same verbal or motor response; loss of ability to carry out familiar movements; intermittent lapse of an assumed posture; lack of muscular co-ordination; irregularity of muscular action; difficulty with attention and concentration;

visual disturbances; poor calculation; difficulty with abstraction; seizures; delusions; hallucinations; depression; psychosis; and sometimes suicide. (151)

These symptoms usually, but do not always, improve after aluminum is removed from the body by treatment with chelating drugs. (101, 163, 152)

Not all of the dialysis patients who receive the same high doses of aluminum develop DES, nor does it usually occur in adults who take large oral doses of aluminum-containing antacids but are not undergoing dialysis. (163, 152)

There is no evidence that patients with DES will go on to develop Alzheimer's Disease, possibly because they do not live long enough to do so. (152) Although brain aluminum content is greatly elevated in DES patients, they do not develop the neurofibrillary degeneration which is a marker of Alzheimer's Disease. (163)

In animal studies, there is evidence of encephalopathy being induced in cats whose brains were directly injected with aluminum chloride or lactate (21) and in rabbits after central nervous system exposure to aluminum. (161)

However, the changes in the brains of humans and animals with aluminum-induced encephalopathy are not identical to the changes which occur in Alzheimer's Disease (35, 122, 124, 29, 164) (explained further below).

In 1921, Spofforth reported a single case of occupational "aluminum poisoning" which caused loss of memory, tremor, jerking movements and impaired co-ordination. The patient also suffered from vomiting, constipation and incontinence of urine. (148)

An unusual case of encephalopathy in an aluminum powder factory worker was reported in 1962. (105) At autopsy, the worker's liver contained 122 times the normal amount of aluminum and his lung and brain aluminum levels were 18 and 17 times normal respectively, but there were no characteristics of Alzheimer's Disease in his brain. No other cases of neurological signs or symptoms were found at the time in that workforce. In the absence of any other explanation, the authors concluded that "it seems possible that the encephalopathy was due to aluminum intoxication."

Despite large numbers of aluminum-exposed workers, there is little evidence that an encephalopathy similar to DES occurs after occupational exposure to aluminum.

A 1977 NIOSH occupational hazard survey estimated that there were about 3 million workers with occupational exposure to aluminum compounds in the United States alone (112) (comparable Canadian statistics are not available (11)), yet there is little evidence of encephalopathy occurring after occupational exposure to aluminum.

Since a disease must have a probable connection to occupation, on the basis of the present evidence the Panel can only conclude that, in the industrial setting, aluminum does not cause a toxic encephalopathy similar to DES.

Aluminum and Alzheimer's Disease

Alois Alzheimer first described this progressive brain disorder in 1907. It occurs in middle or late life and involves a progressive deterioration of memory, disorientation and eventually dementia. (104, 25, 152) Some regard it as a process of accelerated aging. (97)

Since aluminum is widely believed to be the cause of DES, it is important to know that Alzheimer's Disease and DES are very different diseases with different symptoms and courses.

Since aluminum is widely believed to be the cause of DES, it is important to know that Alzheimer's Disease and DES are very different diseases with different symptoms and courses. (24, 26, 79, 80, 29, 152, 164)

The features of Alzheimer's Disease are progressive memory loss, disorientation and mood disturbances. (152) The main the features of DES are speech difficulties, loss of coordination, a characteristic tremor, involuntary shaking, contractions of groups of muscles, loss of memory and personality changes (1, 151) among others, which are described in detail above.

The diagnosis of Alzheimer's Disease is made when neurons with neurofibrillary tangles and numerous characteristic senile (or neuritic) plaques are found in the brains of these patients at autopsy. (35, 163) Much smaller numbers of plaques occur in normal, healthy people as they age. (13, 48, 152)

Alzheimer's plaques contain deposits of amyloid protein and paired-helical filaments (pairs of filaments wound around one another), both of which have been found to contain aluminum. (163)

In contrast to the paired helical filaments which occur in Alzheimer's Disease, the neurofibrillary tangles which have been induced by aluminum in animals have structurally normal "straight" filaments. (163, 29, 48) In addition, aluminum-exposed animals do not develop the senile plaques which are a feature of Alzheimer's Disease. (163)

There were no Alzheimer-type neurofibrillary tangles in the brains of patients who died of DES despite the elevated concentrations of aluminum in some parts of their brains. (163)

Dr. D. R. Crapper McLachlan states that "Aluminum induces neither the paired helical filament configuration found in Alzheimer-type neurofibrillary tangles nor the formation of senile plaques with amyloid cores." (31, 29) However, he and other researchers

believe that aluminum plays some important but so far unidentified role in the disease (22, 121, 86, 48, 29, 31), while others do not. (4, 85, 163, 79, 80, 164)

Clinical evidence suggests that aluminum may play some role in Alzheimer's Disease because some researchers have found abnormally high levels of aluminum in the brains of these patients at autopsy. (22, 121, 122, 123, 13, 124, 86, 48, 30, 31) Others have not found the same high brain aluminum content in Alzheimer's Disease patients compared to controls. (85, 81, 163)

High levels of aluminum and calcium have also been found in the brains of victims of Amyotrophic Lateral Sclerosis and Parkinson's Disease but again, the microscopic appearance is not identical to Alzheimer's Disease. (168, 122, 163)

One recent clinical trial involving 48 subjects suggested that the progression of Alzheimer's Disease had been slowed after levels of aluminum in the body were reduced by injection of a chelating agent. (30) However, as discussed below, chelation therapy also removes iron and alters the body's chemical and hormonal balance.

In the animal study of encephalopathy (mentioned above) injection of aluminum into the brains of rabbits produced a fatal encephalopathy and neurofibrillary degeneration similar but, again, not identical to that seen in Alzheimer's Disease. The authors proposed that Alzheimer's Disease involves a defect in the olfactory mucosa which allows excessive aluminum into the brain. (125)

Aluminum may accumulate in the brains of Alzheimer's Disease victims because the disease causes a defect in the normally protective blood-brain barrier which makes the brain a willing host for aluminum deposition.

Researchers do not agree about whether aluminum is one of the factors which causes Alzheimer's Disease or whether its accumulation in the brain is a secondary effect of the disease. Aluminum may accumulate in the brain because the disease causes a defect in the normally protective blood-brain barrier which makes the brain a willing host for aluminum deposition. (35, 24, 26, 4, 27, 28, 124, 85, 79, 53)

Some studies suggest a relationship between the incidence of Alzheimer's Disease and aluminum levels in local drinking water (156, 99, 116) but other researchers consider that suggestion "premature" (136) and "unwise." (46) Another study did not find such a relationship. (159)

While the cause of Alzheimer's Disease remains unknown, aging, head injury, family history, genetic factors and thyroid disease have been linked. (75, 76, 77, 57, 113, 72, 7, 67, 127, 66)

There is increasing evidence that an altered protein, called "amyloid," is encoded by a gene on chromosome 21 in some late-onset familial Alzheimer's Disease patients. (74, 62, 137, 167, 63) As discussed above, amyloid is one of the components of senile plaques

which, in large enough numbers at autopsy, are one of the diagnostic features of Alzheimer's Disease. Victims of Down's Syndrome, who have an extra copy of chromosome 21, produce more amyloid and develop more plaques than do people without the disease. Down's Syndrome victims almost invariably go on to develop the pathological changes of Alzheimer's Disease if they live beyond age forty. (75, 35, 162, 137, 167) Other potential genetic causes of Alzheimer's Disease are currently being researched.

Anecdotal reports of dizziness, headaches, nausea, memory loss and irritability have been made by the aircraft workers. The Panel's consultant Neurologist, Dr. J. Turnbull, advises that, other than memory loss which has many potential causes, the symptoms reported by these workers are not the symptoms of Alzheimer's Disease nor of DES but are almost exactly the symptoms seen in patients with chronic solvent exposure. (166, 8, 151)

Obviously, the Panel is not in a position to draw any conclusions about the cause(s) of the symptoms of dizziness, headaches, nausea, memory loss and irritability, but it must acknowledge that high levels of solvents in the work environment at McDonnell Douglas have been identified by the Ministry of Labour. For example, air test results showed that levels of trichloroethane were as high as 6074, 6027 and 5236 p.p.m. in 1985 when the time-weighted average guideline was 350 p.p.m. (109)

***N**either the occupational surveys nor the medical literature provide evidence that Alzheimer's Disease has a general occupational link.*

In summary, neither the occupational surveys (discussed above) nor the medical literature provide evidence that Alzheimer's Disease has a general occupational link.

Occupational Aluminum Exposure and Cognitive Deficits or Neurologic Effects

As discussed above, very high aluminum exposure is believed to have caused neurological and cognitive deficits in some dialysis patients, resulting in problems with speech, concentration and muscle control, a condition which is known as DES.

The Panel also notes that cognitive deficits were found in dialysis patients without extremely high blood aluminum levels and without overt DES (6), although it has not been established that those deficits were caused by aluminum. (154)

***T**he Panel has funded the first phase of a follow-up study of the effects of high levels of aluminum exposure on northern Ontario miners.*

Between 1944 and 1979, northern Ontario miners were deliberately exposed to high levels of an aluminum powder (353 milligrams/cubic metre of air), known as "McIntyre Powder", which contained metallic aluminum and aluminum oxide, in an attempt to prevent the occurrence of silicosis. (42) An epidemiological survey of a small sample of those miners suggests that they may have suffered some cognitive effects as a result of that aluminum exposure. (126) This was a preliminary survey only and some of the

methodology used has since been criticized. (153) However, these could be very important findings, particularly when considered in light of the studies discussed below. The Panel has funded the first phase of a more detailed study of the aluminum-exposed miners in an attempt to repeat those findings.

A Yugoslavian study of 87 aluminum foundry workers found that their "changes in psychomotor and intellectual abilities could be a consequence of the long-lasting toxic effects of aluminum." (78)

A Swedish study of 65 welders found that "Welders exposed to aluminum, lead or manganese for a long period had more neuropsychiatric symptoms than welders not exposed to these metals." The authors attributed these findings to metal welding fumes, but not specifically to aluminum. (142)

Three cases of neurologic disorder in potroom workers occurred "in one of the largest aluminum production plants in the United States." Those workers had been heavily exposed to particulate alumina, fluorides including hydrogen, calcium and lithium, hydrogen sulfide, sulfur oxides, polycyclic aromatic hydrocarbons, volatile emissions of coal tar pitch, carbon monoxide, carbon dioxide, heat, noise and strong direct-current electrical fields. None of the three cases had evidence of other diseases reported to afflict workers in the aluminum production industry and none had the clinical features of DES, osteomalacia nor anaemia which have been linked to aluminum. They had some cognitive impairment but not the same impairment seen in DES. The authors attributed these effects to occupational exposure but were not able to specify from which substance(s). (96)

The OWHC provided anecdotal evidence to support its argument that at least 10% of the 375 McDonnell Douglas workers whose blood aluminum levels were higher than normal suffer from noticeable memory loss. (68) The Panel has not yet received the requested documentation of these findings.

The Union states that some workers from both aircraft plants have filed aluminum-related claims through their sickness and accident insurance plan and that numerous claims have also been filed with the WCB. All those WCB claims which were based on aluminum levels without other symptoms or diagnoses have been denied. Several of their claims related to solvent exposures have been allowed by the WCB. (114, 52)

The Panel's Findings Regarding Occupational Aluminum Exposure

The Panel accepts that there is some epidemiological and anecdotal evidence to suggest that high levels of occupational aluminum exposure might lead to cognitive or neurologic effects. Is that evidence sufficient to conclude that a probable relationship exists between aluminum exposure and disease?

In the Panel's opinion, the *Act* does not require scientifically conclusive evidence to establish the existence of an industrial disease. It is only necessary to find a **probable** connection between a process and/or exposure to a substance and the development of a disease. However, that probable connection can only be made when an adequate level of evidence exists.

In weighing this evidence, the Panel notes that the medical and epidemiological evidence is preliminary. There is an insufficient number of studies to establish consistent results. The problem is that exposure to aluminum is usually accompanied by exposure to other potentially toxic substances and therefore it has not been possible so far for aluminum to be identified as the causal agent of any occupational disease. In addition, the anecdotal reports have not been supported by any documentation. Overall, the evidence currently available to the Panel is inadequate to allow the Panel to describe occupational exposure to aluminum as a probable factor in the development of cognitive or neurologic deficits, or Alzheimer's Disease. Therefore they cannot currently be described as aluminum-related industrial diseases.

However, the Panel recognizes that a lack of evidence does not mean that a relationship does not exist. It only means that medical science cannot yet answer these questions. Clearly, further research is needed in order to resolve the significant uncertainties about aluminum and to answer the important concerns of workers. Further research is already being funded by this Panel, as discussed below.

If evidence can be obtained which identifies a disease that has a probable connection to workplace exposure to aluminum, this Panel will promptly reconsider its opinion and these interim findings.

Further Study of Health Effects from Aluminum Exposure

The Panel's Progress Report of October 20, 1989 identified the need for further investigations of the health effects of aluminum and other workplace exposures in aircraft workers. (45) Accordingly, the Panel has considered funding a proposal entitled "A Study of Aircraft Workers Exposed to Aluminum." (89)

It has heard extensive critiques of that study from the Union (15, 37, 38) and management (39, 100) as well as from numerous medical and scientific experts (17, 143, 55, 151), the Ministry of Labour (102) and the WCB. (51)

After considering all of those opinions, the Panel has concluded that this proposal will not distinguish between the effects of aluminum and the effects of solvents because it cannot adequately control for solvent exposure. Therefore, it cannot adequately answer the workers' questions about the effects of aluminum exposure and other workplace exposures on their health because it cannot distinguish between potential causes of ill health. For these reasons, the Panel will not proceed with that study proposal.

However, the Panel has already funded the first phase of a follow-up study of the effects of aluminum exposure on northern Ontario miners, who were exposed to high levels of aluminum in the form of "McIntyre Powder" from 1944 to 1979. (126) These workers were not also exposed to the potentially confounding effects of solvents.

The Panel will consider funding further phases of this study once it has reviewed the first-phase findings.

2) ***Are serum aluminum levels a good indicator for predicting the occupational health risks? Are urine aluminum levels or other measuring methods useful as indicators for predicting occupational health risks?***

Background

Much of the previous medical research on aluminum has studied aluminum levels in blood and urine because these are the easiest samples to obtain. (5) Aluminum in bone has been studied in research about osteomalacia, a condition marked by softening of the bones due to impaired mineralization. Brain aluminum levels have been studied at autopsy in research about Alzheimer's Disease and other dementias.

The only effective route of elimination is through urine.

It is known that aluminum exposure may be reflected in increased blood and urine levels, at least temporarily. The only effective route of elimination is through urine. (134, 5)

The Panel has noted several examples of wide variations in aluminum levels which occur because of differences in diet, intake of medication, state of health and the type of aluminum involved (32):

Wide variations in blood and urine aluminum levels occur because of differences in diet, intake of medication, state of health and the type of aluminum involved.

- Certain types of aluminum appear to penetrate the body's protective barriers much more than do others. Aluminum maltol, for example, is a sugar-based additive in many processed foods. When fed to rabbits, it was found to increase urine levels by more than 200 times and to raise brain aluminum levels by three times (87);
- Tea leaves are grown on one of the few plants that naturally accumulate aluminum. Brewed tea typically contains 2 to 6 mg/litre of aluminum and ingestion markedly raises urine aluminum levels (56);
- Normal urine levels increase from 4 to 50 times when antacids are being taken (84);
- Coated aspirin contains about 12–17% aluminum (71);
- Iron deficiency and parathyroid hormone promote aluminum absorption (145);
- Ingesting dill pickles, drinks from aluminum cans and acidic foods cooked in aluminum containers raises blood aluminum levels (32);
- Blood aluminum levels will increase by 2–1/2 to 5 times when a person takes antacids in combination with acidic foods such as orange juice (88, 84);
- Milk of Magnesia is 50% aluminum. One non-occupationally exposed patient taking Milk of Magnesia had a blood aluminum level of 1300 nmol/litre (157);
- Dr. J. A. Fenton, staff physician for McDonnell Douglas, has provided the Panel with laboratory reports which indicate that he had 55 nmol/litre of aluminum in his blood prior to taking 15 cc of Maalox and this level nearly doubled (to 95 nmol) within 12 hours. (103)

These are only a few examples which are given to illustrate the fact that people without occupational exposure to aluminum can have blood or urine levels which are considerably above "normal" levels.

The Evidence about Blood Aluminum Levels

It must be noted that some researchers maintain that blood aluminum cannot be measured accurately. (9) Others disagree and have cited the following normal levels of aluminum in the blood of non-occupationally exposed persons:

level	*author*
average of about 241 nmol/litre	Kaehny et al., 1977
between 74 and 519 nmol/litre	Leung and Henderson, 1982
means between 78–1556 nmol/litre	Savory et al., 1983
about 371 nmol/litre	Henderson, 1992

These variations have been attributed to differences in methods of analyzing samples as well as differences in diet, intake of medication, state of health and the type of aluminum involved. (32, 73)

Whether significant or not, researchers report that Alzheimer's Disease patients have normal blood aluminum levels. (23, 24)

Blood aluminum levels are not considered to reflect body burden.

Blood aluminum levels and body burden
Blood aluminum levels are not considered to reflect body burden. (5, 152)

In dialysis patients, aluminum in bone has been found to correspond with the total body burden but blood levels did not. (160)

Animal research leads to the same conclusion. After five months of low-level exposure to aluminum oxide dust, rabbits' brains accumulated two and a half times the aluminum levels found in the non-exposed and lung levels were 158 times higher, but blood levels were only slightly raised. Again, these authors concluded that blood levels do not indicate body burden of aluminum. (131)

Blood aluminum levels are not considered useful for occupational monitoring.

Blood aluminum levels in occupational monitoring
It is established that aluminum is cleared relatively quickly from the blood and excreted in urine when people have normal kidney function (5, 152) so blood levels are not considered useful for occupational monitoring. (65)

Several researchers who have studied blood aluminum levels in occupationally exposed workers also concluded that blood levels are not reliable for occupational monitoring. (155, 138, 115, 141, 33)

The Ontario Ministry of Labour has issued a Circular for Health and Safety Support Service Branch Consultants, dated July 13, 1988, which states:

> The normal level of aluminum in serum has been reported to be 372 nmol/L. Work practices should be reviewed when a worker serum aluminum level is at or above 744 nmol/L. There is no support in the literature for a serum aluminum level that indicates toxicity in individuals with normal renal [kidney] function. (107)

The Panel has been advised that this level was chosen arbitrarily in response to the workers' complaints. (157) Obviously, this is not the appropriate way to choose a standard for acceptable blood aluminum levels.

Dr. Donald R. Crapper McLachlan is a Toronto Neurologist who has conducted much of the leading research into aluminum health effects. He and several of his colleagues attended the Panel's

meeting on December 11, 1991 and provided considerable information and expert advice. They made clear that blood aluminum levels cannot be used to predict bone, liver or brain aluminum levels. They also confirmed that the relationship between blood aluminum levels and the probability of cognitive deficit is unknown. This was reiterated by Neurologist Dr. J. Turnbull, who also provided expert advice to the Panel at that meeting.

For those reasons, Dr. Crapper McLachlan has advised the IDSP that he would not make decisions about when to remove people who are not reporting cognitive symptoms from workplace aluminum exposure based upon their blood aluminum levels because blood levels are not considered predictors of danger to health. (33) He also said:

> Perhaps the most rational approach would be to argue that an individual, in whom the most probable diagnosis for either cognitive or motor control deficits was most likely related to aluminum, should be removed from the workplace if the serum value exceeds 10 micrograms [370 nmol] per litre. However, the long-term risk to those individuals having an elevated blood aluminum level without neurological signs is uncertain and requires further systemic investigation. (34)

In all of its research and consultations with the parties, the Panel has not been able to find any cases of cognitive deficits nor of other adverse health effects which were diagnosed as related to aluminum in the workers from either of these two aircraft plants.

In summary, none of the numerous medical experts consulted by the Panel was able to identify a blood aluminum level which indicates danger from further exposure when no symptoms are being reported. Since aluminum is quickly cleared from the blood and either excreted in urine or deposited in bone or other tissue, low blood levels of aluminum in exposed persons can mean that aluminum is either being excreted or retained. Therefore low blood aluminum levels do not necessarily indicate low risk to health.

The Evidence about Urine Aluminum Levels

The following have been cited as normal levels of aluminum in the urine of non-occupationally exposed persons:

level	*author*
median of about 148 nmol/litre	Sjögren et al., 1983
average of 155 nmol/litre	Mussi et al., 1984
average of 279 nmol/litre	Hosovski et al., 1990
less than 370 nmol/litre	Leung and Henderson, 1982
average of 491 nmol/litre	Kaehny et al., 1977

There is nothing in the literature which suggests a "safe" level of aluminum in urine.

Urine aluminum levels are not affected by gender, age or smoking (138) but, like blood levels, can be markedly affected by diet and intake of medication.

Urine aluminum levels and body burden
The Panel's investigations found nothing in the literature to suggest that urine aluminum levels reflect body burden. This was confirmed by the medical experts who attended the Panel's meeting on December 11, 1991. (32, 152)

Urine aluminum levels in occupational monitoring
While urine levels more often correlate with exposure than do blood levels, urine measurements are also not reliable because there are great variations between individuals. (141) Variations may also be caused by contamination of samples and differences in analyzing methods. (32, 73)

Some studies suggest that urinary aluminum levels are proportionate to the amount of aluminum exposure (138, 115, 141, 78), but another study found no significant increase in urine levels after low levels of exposure, indicating that aluminum had accumulated in the body rather than having been excreted. (130)

No proportionate increase in urinary aluminum levels was found in a study of long-term exposed workers. (95) Other studies indicate a much slower rate of excretion after long-term exposure (140, 50), again suggesting that some aluminum had been retained in the body.

"High" urine levels suggest that the body's protective mechanisms are efficiently clearing aluminum.

These findings were reiterated by all the doctors who were consulted by the Panel. Low urine levels of aluminum in exposed people might mean that aluminum is being retained in the body, so low urine aluminum levels do not necessarily indicate low risk to health. In fact, the Panel is advised that high urine levels suggest that the body's protective mechanisms are efficiently clearing aluminum. (152)

The Panel's Finding Regarding Blood and Urine Aluminum Levels

All of the medical experts consulted by the Panel concurred with the majority of the medical literature and stated that neither blood nor urine levels consistently reflect exposure or absorption. Without exception, the Panel has been advised that neither blood nor urine levels indicate the total body burden of aluminum.

On this strong evidence, the Panel must find that neither blood nor urine levels of aluminum indicate the amount of aluminum which is being retained by the body, nor where it might accumulate, nor whether there is any danger to health from occupational exposure. The evidence shows that blood and urine aluminum levels are clearly inadequate measurements for indicating or predicting danger to health from aluminum.

Neither blood nor urine levels indicate a "medical condition" which may be a precursor to an industrial disease.

Since neither blood nor urine aluminum levels indicate an abnormal state of health, neither can be considered a "medical condition" which may be a precursor to an industrial disease.

The Evidence about Other Measuring Methods

There are other methods which may be capable of assessing levels of aluminum in the body, such as bone biopsy, autopsy and the use of chelating drugs:

1) So far, the most reliable tissue for assessing body burden of aluminum is bone. (5) Alfrey reports that bone aluminum levels bear no relationship to blood aluminum levels. (3) Malluche and Faugere speculate that avid uptake of aluminum by bone may protect against aluminum accumulation in the brain. (98)

These are not yet conclusive findings but, in any event, surgical bone biopsy is far too invasive a method to propose for medical monitoring of apparently healthy workers in order to satisfy the Workers' Compensation Board that it should remove workers from further aluminum exposure;

2) Brain aluminum content can, so far, only be determined by surgical biopsy or at autopsy (152);

3) Chelation therapy, which involves a series of injections of a drug known as "desferrioxamine," mobilizes accumulated aluminum.

Dr. Crapper McLachlan and colleagues have advised the Panel that more could be learned about whether and how blood aluminum levels correlate with aluminum exposure and body burden by conducting desferrioxamine challenge tests on the aircraft workers, then measuring their blood aluminum levels.

However, other researchers, and the drug's manufacturer, have described potentially serious side effects when several chelation therapy treatments are given, such as reported cases of potentially fatal blood infections, temporary and permanent damage to hearing and vision, allergic or hypersensitivity reactions, nausea, diarrhea and lowered blood pressure. (93, 64, 118, 119, 12, 98, 30, 152) Worsening of encephalopathy and of seizures has been reported even at low doses. (101, 94)

In addition to removing aluminum, chelation treatments remove iron from all tissues and disturb the blood concentration of parathyroid hormone which is necessary for normal health. (44) When used in cases of lead toxicity, it may even cause increased absorption of lead. (93) Finally, some studies suggest that some of the drug may accumulate in the body. (43)

Perhaps because the "half-life" of desferrioxamine is about 90 minutes (26) (i.e., it takes about 90 minutes for half of the drug to be cleared from blood), it does not remain effective long enough to inspire confidence that blood levels after chelation indicate body burden. (5) Moreover, it is not known whether body burden reflects danger to health. (5, 152)

Under these circumstances, the Panel is not prepared to recommend conducting experiments on apparently healthy workers who are not reporting symptoms when doing so could cause them illness and which will not provide reliable answers about their health in any event.

There is currently no acceptable tool which is capable of reliably determining whether, how much or where aluminum may be accumulating in the body.

In summary, there is currently no acceptable tool which is capable of reliably determining whether, how much or where aluminum may be accumulating in the body. However, the Panel is anxious to find a responsible way to protect workers from risk without unnecessarily alarming workers who are not at risk and so it is investigating current research into other methods of assessing body burden of aluminum.

The Panel is monitoring research into applying a new monitoring method to aluminum, known a "neutron activation analysis."

The Panel is encouraged to learn about research into a new testing method, known as "neutron activation analysis", which has already been shown to reliably indicate body burden of lead and cadmium. (18) Dr. David Chettle and colleagues at McMaster University are currently working on refining that technique to make it safer and more sensitive, and on ways to apply it to aluminum and to mercury. (18, 19) The Panel is cautioned, however, that body burden does not necessarily predict any potential risk of cognitive effects. (154)

While that measurement tool is being developed, the Panel asks that the Union and the employers help it find the needed answers by continuing to collect and provide evidence of any symptoms and/or disability which might be related to occupational aluminum exposure, or to any other exposures which occur in the aircraft industry. The Panel undertakes to continue investigating the issue and this important new technique and will make further findings known to the parties as soon as more information becomes available.

3) Under what conditions should a worker be removed from occupational exposure to aluminum?

Section 1 (1) (n) (iii) of the *Workers' Compensation Act* states that an "industrial disease" includes:

> a medical condition that in the opinion of the Board requires a worker to be removed either temporarily or permanently from exposure to a substance because the condition may be a precursor to an industrial disease.

The purpose of the *Act* is to provide for payment after a worker becomes ill. The *Act* does not have a general safety mandate. The precursor clause is one of the few legislative provisions that allows the WCB to be proactive and protective in terms of the worker's health and safety.

There must be a "medical condition" which is connected to work or occupational exposure to a substance, but it need not be disabling.

The Panel's Finding regarding the "precursor clause"

In the Panel's reading of this provision, it is not necessary that there be a disabling feature to the "medical condition." If disability were necessary, it would mean that an industrial disease had already been established and the preventative feature of the legislation would be lost. It is of interest to note that Workers' Compensation Appeals Tribunal decision #157/91 confirms the need to identify a medical condition and also concluded that it was not necessary for disabling consequences to flow from the medical condition.

However, it is necessary that the medical condition be a condition which "may be a precursor to an industrial disease." In the Panel's view, regardless of whether or not blood and urine levels are "medical conditions", they do not constitute precursors to any known aluminum-related industrial disease. Nor are there any other acceptable methods for diagnosing potential precursors to aluminum-related industrial disease. Hence, the Panel is unable to identify conditions under which workers should be removed from occupational exposure to aluminum.

Summary of the Panel's Findings

1) The evidence currently available is inadequate to allow the Panel to conclude that occupational aluminum exposure causes neurological health effects.

2) Neither blood nor urine aluminum levels are good indicators for predicting occupational health risks, nor for estimating body burden of aluminum. The other measuring methods which are currently available are too invasive to be considered for occupational monitoring. There is currently no evidence that body burden of aluminum reflects danger to health.

3) The available evidence is inadequate to allow the Panel to identify conditions under which a worker should be removed from occupational exposure to aluminum.

4) Since the available evidence is inadequate to identify adverse health effects from occupational aluminum exposure, the Panel is not able to recommend criteria for the adjudication of claims at this time.

Summary of the Panel's Recommendations

Since some preliminary studies suggest that aluminum could cause some health effects, further research is recommended.

The Panel recommends that a study of health effects in aircraft workers be conducted, subject to the parties' agreement to participate and the availability of sufficient funding.

Further Panel Activity

The Panel has funded the first phase of a follow-up study of cognitive effects in northern Ontario miners exposed to aluminum and will consider funding further phases of this study once the initial findings have been reviewed by the Panel.

For the reasons outlined above, the Panel will not proceed with the proposed study entitled "Proposal: A Study of Aircraft Workers Exposed to Aluminum."

The Panel will continue to monitor research into the application of neutron activation analysis to aluminum.

The Panel undertakes to continue to monitor all research about potential health effects of aluminum.

The Panel again requests that the Union and management continue to provide any and all evidence of health effects in aircraft workers which becomes available to them. Once further scientific or medical evidence is available, the Panel will promptly reconsider these findings.

Industrial Disease Standards Panel
May, 1992

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